ABSTRACTS OF CARDIOLOGY

Neosynephrine in Treatment of Paroxysmal Supraventricular Tachycardia. W. B. YOUMANS, M. J. GOODMAN, and J. GOULD. *Amer. Heart J.*, **37**, 359–373, March, 1949.

"Neosynephrine" differs from adrenaline in that the molecule has a hydrogen atom instead of a hydroxyl group in the *para* position on the benzene ring. It causes marked hypertension and peripheral vasoconstriction. Its administration is followed by a sinus-inhibition bradycardia. In animals this inhibition may result in a ventricular escape.

The authors studied 19 attacks of paroxysmal supraventricular tachycardia occurring in 9 patients. Up to 2 mg. of neosynephrine was given intravenously during an attack. In only 2 cases, both after major operations, did the rhythm fail to revert to normal. Reversion occurred within 35 to 70 seconds of injection. Unpleasant side effects were few. One case of paroxysmal ventricular tachycardia failed to respond. *A. S. Dixon*

Angiocardiography in Congenital Heart Disease of Cyanotic Type with Pulmonic Stenosis or Atresia. I. Observations on the Tetralogy of Fallot and "Pseudo-Truncus Arteriosus." R. N. COOLEY, H. T. BAHNSON, and C. R. HANLON. *Radiology*, **52**, 329–346, March, 1949.

The authors used angiocardiography in 75 cases of congenital heart disease of the cyanotic type. They employed a modified Robb and Steinberg technique for the injection. The contrast medium was a 70% solution of "diodrast" injected into the basilic vein of the left arm or into the external jugular vein in small children; 140 injections were made without serious reaction.

Nine cases are reported in detail. Seven had the tetralogy of Fallot and 2 had a truncus arteriosus. All were subsequently operated upon, and in one case post-mortem studies were available. An overriding aorta could be demonstrated in all cases. An interventricular septal defect may sometimes be directly demonstrated. Direct visualization of the pulmonary conus is so difficult as to be an untrustworthy guide, but strong presumptive evidence of stenosis may often be obtained. It is not possible by angiocardiography alone to distinguish between pulmonary atresia and a high degree of stenosis. The presence and character of the pulmonary arteries may be reliably determined. Failure to demonstrate the proximal position of the pulmonary artery, however, does not rule out the possibility of a successful anastomosis. *A. Orley*

Cardiac Catheterization in Congenital Heart Disease. By A. COURNAND, M.D., JANET S. BALDWIN, M.D., and A. HIMMELSTEIN. Pp. 108: illustrated. $4.00 or £1 2*s*. New York; The Commonwealth Fund. London: Geoffrey Cumberlege, 1949.

The practical and pioneer contributions of Cournand and his colleagues in applying the technique of cardiac catheterization are well known. This monograph is a detailed study of some cases of congenital heart disease. The first part gives a clear and concise account of the staffing, equipping and technical methods involved in catheterizing the right heart, with a note on its complications. Radiographic observations and tracings of blood pressure recordings taken during catheterization are also illustrated, and formulæ given for calculating blood flow and shunts. The second part of the book consists of a useful selection of seventeen clinical cases illustrating the commoner types of a cyanotic heart disease in which this diagnostic technique has been used. The results are analysed and interpreted in simple language which provides a valuable summary of the scope of this procedure. The volume should serve a useful purpose in emphasizing the highly specialized nature of the investigation of cardiovascular malformations by catheterization. The authors acknowledge these limitations, but in presenting their data so graphically have undoubtedly done service to clinical cardiology. *J. L. Lovibond*

Aspiration of Blood from Pericardium in Treatment of Acute Cardiac Tamponade after Injury. Further Experience, with Report of Cases. M. M. RAVITCH and A. BLALOCK. *Archives of Surgery*, **58**, 463–477, April, 1949. 4 figs.

Despite the lowered operative mortality rate during the last 5 years in the treatment of wounds of the heart, the authors reaffirm their opinion, first expressed in 1943, giving preference to the more conservative pericardial aspiration for hæmopericardium causing tamponade, and reserving cardiorrhaphy for those patients in whom bleeding continues or is resumed after aspiration.

In a further series of 10 cases manifesting signs of tamponade careful records are supplemented by illustrative charts of progress. Eight patients were successfully treated by aspiration, one was subjected to immediate cardiorrhaphy, and the other to operation after two aspirations. All survived without complications.

C. A. Jackson

Clinical Auscultation of the Heart. By S. A. LEVINE, and W. P. HARVEY, (W. B. Saunders Company—Philadelphia and London). Price 32*s.* 6*d.* Pages 327.

The authors have been impressed by the reluctance of many modern clinicians to make full use of the stethoscope, and find the availability of more complicated, and perhaps more impressive apparatus for the investigation of cardiovascular disorders tending to supplant the simpler method of auscultation by the stethoscope. Auscultation may be a small part of the total assessment of cardiac function yet it is a very important and often a very simple one. The value of this book lies not so much in its lucid exposition of phonocardiology, amply illustrated by excellent photographic reproductions, as in the co-relation of these instrumentally obtained records with clinical auscultatory findings. The work contains much common sense, practical observation, and good teaching that will profit all clinicians; its appearance is the more acceptable at a time when the study of heart sounds and murmurs is the subject of renewed interest.

J. L. Lovibond

Coagulation, Thrombosis, and Dicumarol. SHEPARD SHAPIRO, M.D. and MURRAY WEINER B.S.M.S., M.D., Brooklyn Medical Press, New York. 1949. Price $5.50.

The discovery and synthesis of dicumarol by Link and co-workers placed a powerful weapon in clinicians hands to combat thrombosis. The senior author of this monograph was closely associated with Link's group when the question of using this new drug in man was considered; the present work is based largely on the results of their investigations in this field.

The chapters on the relationship between decreased coagulability, low prothrombin, and hæmorrhagic phenomena are valuable. That hæmorrhages may occur when the coagulability of the blood or its prothrombin content are but little altered should be recognized. Furthermore, the control of such drugs needs the closest collaboration between clinician and pathologist. The so-called "prothrombin time" estimation is not a measure of one substance but rather of the behaviour of a complex of reactions occurring simultaneously under more or less standard conditions. The relation between prothrombin activity and vitamin K is very clearly expressed, especially in the presence of liver disease. Salicylates potentiate to a marked degree the lowering of prothrombin by dicumarol.

There is a clear discussion on the dose and control of the drug in which the authors give their reasons for preferring the "intermittent dosage" method by which a dose large enough to guarantee a therapeutically effective prothrombin time prolongation is given at the onset. No more is given until the prothrombin time tends towards the normal. This method is claimed to be safer than the "daily dose" method. Each case must be the subject of individual attention; general rules of dosage are not practicable.

There is an interesting chapter on the estimation of dicumarol in plasma and body fluids. It would appear to be excreted by the large bowel, since the drug does not appear in bile or urine.

The chief criticism of this work is that it is the representation of one group and that the views of other and conflicting schools are hardly represented at all. Furthermore, the authors have apparently completely misunderstood the two-stage prothrombin procedure. This monograph is of a high order in production.

J. G. Humble

Some Effects of Digoxin upon the Heart and Circulation in Man. Digoxin in Chronic Cor Pulmonale. M. I. FERRER, R. M. HARVEY, R. T. CATHCART, C. A. WEBSTER, D. W. RICHARDS, and A. COURNAND. *Circulation*, **1**, 161–186, 1950. 7 figs., 13 refs.

Intravenous administration of digoxin in doses of 1 to 1·5 mg. caused a rise in cardiac output, a rise in pulmonary artery pressure, a fall in right ventricular and diastolic pressure, and a fall in venous filling pressure in 5 cases of chronic anoxic cor pulmonale with the clinical features of congestive heart failure. A parallel study was made on 3 cases of chronic cor pulmonale and 2 cases of pulmonary hypertension in which there was no evidence of heart failure: in these the effect of intravenous digoxin was negligible. The authors feel that digoxin may well have caused these effects by a direct action on the cardiac muscle, but admit that they were unable to exclude a primary vasodilator action.

When, as a result of medical treatment (rest in bed, digitalization, administration of mercurial diuretics to 2 patients, and antispasmodic inhalations) clinical improvement was obvious, there was a marked increase in arterial oxygen saturation, vital capacity, and maximum ventilation, accompanied by a conspicuous reduction in cardiac output, pulmonary artery pressure, and right ventricular diastolic pressure—indeed, the pulmonary artery pressure in 4 of the patients became almost normal.

The reversibility of chronic cor pulmonale is stressed.

Paul Wood

The Cardiac Output in Normal Pregnancy as Determined by the Cournand Right Heart Catheterization Technique. H. F. H. HAMILTON. *J. Obstet. Gynaec. Brit. Emp.*, **56**, 548–552, 1949. 1 fig., 10 refs.

The cardiac output was determined in a series of 32 non-pregnant women in the child-bearing age and in 75 normal women in various stages of pregnancy. The technique of cardiac catheterization used was similar to that used by Cournand in 1945. In 24 normal non-pregnant women the mean cardiac output was 4·5 litres a minute and the cardiac index was 3 litres a minute. There were 68 successful estimations in pregnant women, whose output began to rise between the 10th and 13th weeks and reached its maximum around the 7th month with an average of 5·7 litres a minute. Following this there was a steady fall until the 40th week when the output was 4·6 litres a minute. The oxygen consumption during pregnancy rose steadily to 251 ml. a minute, compared with an initial value of 211 ml. a minute.

The fact that the cardiac output returns to normal

before the onset of labour is of clinical interest. The work of the heart has decreased during the last weeks of pregnancy and by term it is under optimal conditions to deal with the sudden changes which occur throughout labour, particularly in the third stage. The benefit of this to a patient with a diseased heart is obvious.

[It would seem from this article that the present practice of many obstetricians of inducing premature labour in cases of heart disease is not sound physiologically; the outlook for the patient would be better if the pregnancy was allowed to go to term.] *B. G. Spiers*

Effect of Auricular Fibrillation on the Coronary Blood Flow. R. WÉGRIA, R. P. KEATING, H. P. WARD, F. DREYFUSS, C. W. FRANK, and M. R. BLUMENTHAL. *Amer. J. Physiol.*, **160,** 177–182, Jan., 1950. 3 figs.

Direct measurements of the arterial blood pressure and the blood flow in the coronary circulation were made in 16 dogs under sodium barbitone anæsthesia, and auricular fibrillation was induced by electrical stimulation of one of the auricular appendages. At the onset of fibrillation there was a marked fall in blood pressure and coronary flow. Within a few seconds both blood pressure and coronary blood flow returned towards their original levels and, if fibrillation persisted long enough, rose temporarily above initial values. A similar observation was made at the end of fibrillation, the coronary flow especially rising above control level; both values then decreased progressively, the blood pressure being first to reach its control level. The authors discuss the mechanism and significance of the cardiovascular reactions responsible for the observed phenomena.

[From the clinician's point of view the experimental finding that an attack of auricular fibrillation with a high ventricular rate calls for an increase in coronary blood flow is of great importance. It explains the cause of heart failure in a subject with fibrillation and coronary sclerosis in whom an " increase (in coronary flow) may be neither possible nor sufficient to meet the demands."] *A. I. Suchett-Kaye*

Treatment of Paroxysmal Supraventricular Tachycardia with Lanatoside C. J. G. BARROW. *Ann. intern. Med.*, **32,** 116–119, Jan., 1950.

Lanatoside C is, in the author's opinion, the most effective drug for treatment of an acute attack of paroxysmal supraventricular tachycardia (although it may have to give place to " neosynephrine "). It is non-toxic and quite safe, and is stated to be highly efficacious quickly and almost invariably. It must never be given without electrocardiographic identification of the arrhythmia, and it is contra-indicated in any form of ventricular rhythm. Furthermore, it has little value as a preventive, being far inferior to quinine or digitalis in that respect. The dose is 1 mg. intravenously, repeated if necessary. *G. F. Walker*

Atrophy of the Heart: A Correlative Study of Eighty-Five Proved Cases. H. K. HELLERSTEIN and D. SANTIAGO-STEVENSON. *Circulation*, **1,** 93–126, Jan., 1950. 17 figs., bibliography.

Cardiac Changes in Childhood Tuberculosis. V. M. AFANASEVA. *Arkh. patol.*, No. 4, 52–61, 1949. 5 figs., 19 refs.

Tuberculous myocarditis is usually regarded as an uncommon complication of generalized tuberculosis, and lesions are seldom seen with the naked eye. Non-specific inflammation has been reported much more frequently. In order to re-examine this question, 118 hearts of children dying of tuberculosis were studied with considerable care. Macroscopical tuberculous lesions were found in only 2 cases. Non-specific lesions were common and included perivascular œdema, fibrinoid degeneration and necrosis of vessels, hæmorrhage, and perivascular cellular infiltration. In contrast to some previous findings, there was not a single example in this series of lesions resembling the Aschoff bodies. Fifty cases in which diffuse or focal cellular infiltration was found were studied further by means of serial sections. This revealed a surprisingly high proportion of specific tuberculous lesions: 26 out of the 50, or 22% of the total. Tubercle bacilli were found in only 4 cases. *L. Crome*

The Atrophic Kidney and Hypertension. J. R. KILMAN, E. O. BRADFIELD, and C. M. SIMPSON. *J. Urol*, **62,** 417–425, 1949. 8 refs.

Since Butler reported in 1937 the apparent cure of hypertension by removal of a diseased kidney many cases have been recorded which confirm the fact that nephrectomy may relieve hypertension if the kidney is diseased. The authors report 13 cases of unilateral atrophic pyelonephritis treated by nephrectomy for this purpose. In 7 cases the blood pressure returned to normal and has remained normal for 2 to 9 years. In 5 cases the blood pressure remained the same; 3 of the patients died, after 6 months, 9 months, and 4 years respectively. In one case the blood pressure was still unchanged after 18 months, though the symptoms were relieved. The average age in this group of patients was 28 years. In 6 of the 7 cases with good results the opposite kidney was considered to be hypertrophied, while this was not noted in any case in which results were poor. The authors believe that in patients selected for nephrectomy to relieve hypertension: (1) the affected kidney should show a definite reduction in function; (2) the normal kidney should be hypertrophied, indicating that there is compensation for loss of function in the diseased kidney; (3) the best results can be expected in patients below 45 years of age. *J. E. Semple*

Surgical Treatment of Constrictive Valvular Disease of the Heart. H. G. SMITHY, J. A. BOONE, and J. M. STALLWORTH. *Surg. Gynec. Obstet.*, **90,** 175–192, 1950. 13 figs., 19 refs.

The authors review the history of attempts made to treat valvular stenoses of the heart surgically. They have now operated upon 7 patients for mitral stenosis, and describe the technique of both the transventricular and transauricular procedure which they used. [The original paper should be consulted for the details.] They

discuss the pros and cons of the two approaches, and favour the transventricular method. Two of their patients died. Of the remainder none was made worse, and 4 were improved.

During operation marked arrhythmia of the heart was observed during the displacement of the apex despite the topical application of procaine, but after rest and infiltration of the ventricular muscle with local analgesic further manipulations caused little disturbance. Irregularities were less common when the transauricular route was used.

They discuss the selection of patients for operation, saying that those with evidence of congestive failure should not be operated upon and that the ideal candidates are those suffering from the mechanical effects of the lesion only, without associated myocardial damage. They are of the opinion that in these patients the creation of a valvular incompetence by operation is well borne.

J .R. Belcher

The Retinal Vessels in Hypertension. A. LEATHAM. *Quart. J. Med.*, **18**, 203–215, 1949. 8 figs., 27 refs.

The retinal arteries were studied in 111 cases of hypertension and in 103 controls. Changes in the retinal arteries indicative of hypertension consist of irregularity of the lumen, generalized narrowing (the healthy artery is normally two-thirds the width of its companion vein), pallor of the arteriolar blood column, changes at arteriovenous crossings, and a broadened or exaggerated light reflex. It is suggested that these arterioles should be called hypertensive retinal arterioles.

Changes in the retinal arterioles were much commoner in subjects with a diastolic blood pressure of 110 mm. Hg or over than in those with a raised systolic pressure and a normal diastolic pressure. Radiological evidence of enlargement of the left ventricle and electrocardiographic evidence of left ventricular preponderance were commonly associated with the presence of hypertensive retinal arterioles. *Geoffrey McComas*

The Femoral Pulse Curve in Coarctation of the Aorta. S. BJORK and K. LIEDHOLM. *Acta med. scand.*, **136**, 97–104, 1949. 5 figs.

Femoral and carotid pulse tracings in 3 patients with aortic coarctation were compared with those in 10 normal subjects. The femoral pulse curve of these patients was considered pathognomonic of aortic coarctation in three respects: firstly, the peak of the curve was reached much later than in normal subjects; secondly, this same peak occurred after the end of cardiac systole recorded in the carotid pulse curve, whereas in normal subjects it occurs before the end of systole; thirdly, the descending limb of the curve fell more slowly than in normal subjects and contained no secondary peak. *A. Wynn Williams*

Interatrial Septal Defect. R. S. COSBY and G. C. GRIFFITH. *Amer. Heart J.*, **38**, 80–89, 1949. 14 refs.

This is a short review of the present knowledge of interatrial septal defect and includes tabular presentations of (1) the symptoms and signs in 16 patients with clinical evidence of atrial septal defect, and (2) the pathological findings in a further 19 cases that came to necropsy. No case reports are given. Special attention is called to the sometimes enormous enlargement of the right ventricle. The systolic murmurs which may occur are listed. An associated apical diastolic murmur (in 2 of 16 cases) indicates the presence of mitral stenosis (Lutembacher's syndrome) [but no explanation is given of the frequent pulmonary diastolic murmur (in 7 of 16 cases) associated with marked cardiac enlargement]. Radiological findings are described and their frequency listed. Prominence of the pulmonary artery was constant and sometimes extreme. Pulmonary artery pulsation was present in some cases and was most easily seen on the right side.

The intravenous ether circulation time may give diagnostic information. Some ether may be shunted through the septal defect and, by-passing the lungs, cause a wave of paræsthesiæ over the body, which occurs after the same interval as the arm-to-lung circulation time. Angiocardiography is of little diagnostic value, but cardiac catheterization during fluoroscopy may provide proof of the existence of a defect. *A. S. Dixon*

Defect of the Ventricular Septum. Summary of Twelve Cases and Review of the Literature. A. SELZER. *Arch. intern. Med.*, **84**, 798–823, 1949. 1 fig., bibliography.

A description is given of 12 cases of uncomplicated ventricular septal defect (*maladie de Roger*), together with necropsy findings, and 80 reported cases are reviewed. The following conclusions are reached: (1) The great majority of defects are located between the membranous and the anterior muscular septum, involving both and connecting the sub-aortic area of the left ventricle with the right ventricle immediately in front of the median leaflet of the tricuspid valve. This position is similar to that of the defect in the tetrad of Fallot and in Eisenmenger's complex. In a small minority the defect is lower down the septum. (2) Large defects were usually found in infants and were uncommon in those over 15 years of age. The effect upon the circulation depended upon the size of the defect. If large, the signs were similar to those of atrial septal defect or patent ductus arteriosus, with hypertrophy of the right ventricle and enlargement of the pulmonary artery and its branches. (3) The shunt in an average case of ventricular septal defect is from left to right. It is suggested that those with cyanosis should be regarded as examples of the Eisenmenger complex and that it may be difficult to decide at necropsy whether a minor grade of over-riding of the aorta is present. (4) Much evidence is presented in favour of the view that transposition, Fallot's tetrad, Eisenmenger's complex, and ventricular septal defect have a common ætiology in incomplete detorsion of the primitive tube, ventricular septal defect being the least severe grade. (5) In most cases the classical harsh systolic murmur, with thrill, was present in the 4th interspace. In 5 out of 70 cases no murmur was audible, and in one case a diastolic murmur only. No correlation could be found between the size of the

defect and the type of the murmur. Early diastolic murmurs, possibly due to pulmonary incompetence, were not infrequently present in addition to the harsh systolic bruit. (6) The prognosis depends upon the size of the defect. There is also the well-known high susceptibility to bacterial endocarditis. *C. W. C. Bain*

Studies of Congenital Heart Disease. IV. Uncomplicated Pulmonic Stenosis. J. W. DOW, H. D. LEVINE, M. ELKIN, F. W. HAYNES, H. K. HELLEMS, J. W. WHITTENBERGER, B. G. FERRIS, W. T. GOODALE, W. P. HARVEY, E. C. EPPINGER, and L. DEXTER. *Circulation*, **1**, 267–287, Feb., 1950. 5 figs., 49 refs.

Eight cases of simple pulmonary stenosis are reported with clinical, radiological, and electrocardiographic findings and a description of the circulatory dynamics as determined by means of cardiac catheterization. Angiocardiography was not carried out. There were 6 males and 2 females, aged 11 to 26. None was cyanosed; 3 were symptom-free, and 3 had slight effort intolerance and 2 moderate breathlessness on exertion. They were all normally developed. The systolic thrill and murmur were maximal in the 2nd, 3rd, or 4th left intercostal space. The pulmonary second sound was sometimes normal, but sometimes decreased or increased in intensity. The pulmonary arc was prominent in 6 cases, but the peripheral pulmonary vascular markings were normal. The " P pulmonale " in leads II and III was seen in only one instance. Late electrical activation of the right ventricle due to right ventricular hypertrophy or slight incomplete right bundle-branch block occurred in 5 cases.

In all cases the pulmonary artery pressure was appreciably lower than that in the right ventricle. The arterial oxygen saturation was normal. Samples from the superior vena cava, right auricle, right ventricle, and pulmonary artery were essentially similar. Ventilation, oxygen consumption, arterio-venous oxygen difference, cardiac output, and cardiac indices were similar to those in normal controls.

Responses to effort remained normal at the expense of a considerable rise in right ventricular pressure.

[This article contains a valuable review of the literature.] *Paul Wood*

Congenital Pulmonary Stenosis with Closed Ventricular Septum. K. D. ALLANBY and M. CAMPBELL. *Guy's Hosp. Rep.*, **98**, 18–53, 1949. 29 figs., 25 refs.

It is often stated that pulmonary stenosis with a closed ventricular septum is an uncommon cardiac anomaly: 8 cases are here reported, 7 of them verified at necropsy. The pulmonary stenosis in such cases is of the valvular type, the cusps being fused to form a cupola with an aperture of varying size at its centre. The clinical course is determined by the severity of the stenosis and by the presence or absence of a patent foramen ovale. Ordinarily there are few symptoms other than some degree of dyspnœa until the late teens or early twenties. At this time a mounting pressure in the right side of the heart may make a potentially patent foramen ovale permeable and so allow a shunt from right to left to take place. Cyanosis then appears and gradually intensifies, with increasing disability of the subject. If the foramen ovale is sealed, the patient is acyanotic, but ultimately there may be peripheral cyanosis due to increased utilization of oxygen and possible interference with adequate oxygenation in the lungs. There is a harsh systolic murmur, with or without a thrill, in the 2nd left interspace, while the pulmonary second sound is diminished or absent. The electrocardiogram usually shows right axis deviation, with large, pointed P waves in lead II.

The authors point out that, in this as in other congenital abnormalities such as coarctation, the size of the stenotic pulmonary orifice does not increase in relation to the increasing size of the heart and pulmonary artery, and thus becomes of greater importance as the patient grows. Further, as in other obstructive lesions, the valvular stenosis provides a site for infective processes or increased fibrosis and stenosis.

[This is an important contribution to the study of an abnormality which has so far received little attention, and the original paper should be consulted.]

James W. Brown

Genetic and Environmental Factors in Congenital Heart Disease. M. CAMPBELL. *Quart. J. Med.*, **18**, 379–391, 1949. 21 refs.

After reviewing the literature the author reports personal observations based on over 300 case notes of children with congenital heart disease. About 250 of these were cyanotic and 50 acyanotic; 185 were boys and 155 girls. The only environmental factor of importance found was maternal rubella during the first 2 or 3 months of pregnancy; this was present in 4 cases (1·6% of the cyanotic group). Genetic factors proved to be far more important than environmental ones. This view was supported by the observations that more congenital defects were present amongst the sibs and more distant relatives of these patients than could be expected as the result of chance; moreover, congenital defects in other parts of the body were found in many cases. Several instances of congenital heart disease occurring in a family were encountered, two of them striking; these are described in some detail. Although the causes of congenital heart disease are mainly genetic the risk that the next 2 or 3 children will show any serious congenital defect is so small that it is justifiable to reassure the mother on this account. *A. Schott*

The Differentiation of Mediastinal Tumour and Aneurysm: Value of Angiocardiography. I. STEINBERG and C. T. DOTTER. *Brit. J. Radiol.*, **22**, 567–572, 1949. 12 figs., 9 refs.

The authors stress the importance of accurate preoperative diagnosis of mediastinal masses, mentioning tumours in which pulsation is transmitted and aneurysms that do not pulsate because of the thickness of their walls or the presence of clot. In 1948, angiocardiographic examinations were carried out in 238 cases.

The apparatus used was simple and consisted of a standard stereo cassette changer permitting the taking of 2 films per injection of opaque medium.

G. A. Stevenson

Healed Subacute Bacterial Endocarditis, a New Entity. S. R. KAPLAN, R. H. ROSENMAN, L. N. KATZ, and W. A. BRAMS. *J. Amer. med. Ass.*, **141**, 114–116, 1949. 21 refs.

The authors report their observations on 18 cases of subacute bacterial endocarditis treated by intensive administration of pencillin and followed up for periods varying from 25 to 61 months. The causative organism in all but one case was *Streptococcus viridans*, the exception being a β-hæmolytic streptococcus.

In 6 patients who had dynamically significant aortic insufficiency investigation revealed progressive cardiac failure: two of these died within a year and one of them 3 years later. The remaining 12 patients, none of whom had severe aortic insufficiency, remained well. The authors therefore conclude that gross aortic regurgitation is of grave significance. In their study no correlation was observed between progressive cardiac disability following cure of infection and age, sex, duration of illness before treatment, length of hospital stay, or total penicillin dosage. The authors contend that penicillin, besides being bactericidal, influences the healing process, checks the thrombo-embolic "showers," and thus reduces the incidence of cardiac insufficiency.

S. Karani

Occurrence and Prognostic Significance of Gallop Rhythm. J. FROST. *Acta med. scand.*, **134**, 153–170, 1949. 25 refs.

This investigation was carried out on 6000 adult patients, of whom 57 were examined by phonocardiography. The added sounds in every instance were left-sided and occurred during diastole only. In healthy subjects the physiological third heart sounds were noted less frequently with advancing age.

Gallop rhythm was present in 44% of patients with coronary thrombosis and in 17% of those with arteriosclerosis and hypertension; in all of these the mortality rate was higher and survival time shorter than in those without gallop rhythm. There was also a greater incidence of abnormal electrocardiograms, left bundle-branch block and abnormal T waves in lead I being particularly common. Gallop rhythm was also present in 15% of patients with affections of the aorta and aortic valve; again the prognosis was worse than in those without the added sounds. Gallop rhythm associated with rapid filling appeared to have a worse significance than either the auricular or the summation type.

Gallop rhythm was noted in patients with beri-beri and traumatic heart disease but was seldom present with mitral stenosis or thyrotoxicosis. In cases of congenital heart disease and constrictive pericarditis gallop rhythm was sometimes observed but in these younger patients difficulty was found in differentiating the added sounds from the normal third heart sounds.

The author's conclusion is that gallop rhythm is a sign of lowered functional capacity of the heart, indicative of a bad prognosis; its importance lies in the frequent association with other adverse signs such as cardiac enlargement and serious electrocardiographic abnormalities.

Winston M. L. Turner

A Re-evaluation of Papaverine in the Treatment of Angina Pectoris. A. J. SIMON, N. DOLGIN, A. J. L. SOLWAY, J. HIRSCHMANN, and L. N. KATZ. *J. Lab. clin. Med.*, **34**, 992–997, 1949. 13 refs.

The effect of the oral administration of papaverine on the anginal attacks of 13 patients was observed in a carefully controlled series of experiments. In doses of 400 to 800 mg. daily it did not significantly reduce the number of attacks, except in two cases. In one of these the effect was ascribed to the sedative effect of the drug, and its mode of action in the other case was uncertain. Constipation and drowsiness were noticed as the most common toxic effects. This careful re-evaluation of the use of oral papaverine in the treatment of the anginal syndrome leads the authors to conclude that it is of limited value.

H. E. Holling

Infarction of the Interventricular Septum. D. LITTMANN. *New Engl. J. Med.*, **241**, 89–94, 1949. 5 figs., 1 ref.

The most frequent electrocardiographic sign in infarction of the interventricular septum (apart from those indicative of any cardiac infarction) is that of impaired interventricular conduction or frank bundle branch block. Less commonly the Roesler and Dressler pattern is noted (posterior infarction pattern in standard limb leads with anteroseptal pattern in præcordial leads), and still less frequently the picture of uncomplicated myocardial infarction. In this series of 11 fatal cases, the pattern of simple infarction was always that of infarction of the posterior wall. With combined anterior-wall and septal involvement, bundle branch block was invariably found and was accompanied in 2 cases by A-V dissociation. Bundle branch block is not certain evidence of septal involvement in myocardial infarction and, conversely, septal infarction may occur without definite electrocardiographic evidence. Mortality in septal infarction is high, probably about 70% and, this may well be related to the frequency of arrhythmia and interference with conduction.

T. Semple

Studies on the Circulation of Blood in Man. VI. The Pulmonary Capillary Venous Pressure Pulse in Man. H. LAGERLÖF and L. WERKÖ. *Scand. J. clin. Lab. Invest.*, **1**, 147–161, 1949. 14 figs., 9 refs.

There is no significant anastomosis between the smaller branches of the pulmonary artery, for, if a cardiac catheter is pushed along the pulmonary artery of the living subject until it obstructs a fine branch, fully oxygenated blood may be withdrawn. If the catheter is filled with fluid and attached to a manometer, the pressure recorded is that of the pulmonary capillaries and veins (p.c.v.).

The authors used this method to obtain pulse-pressure

tracings of the p.c.v. in 50 subjects, including normal persons, pregnant women, and patients with hypertension, mitral valve disease, pulmonary emphysema, and auricular septal defect. They conclude that the mean p.c.v. pressure is about 2 mm. Hg higher than the left auricular mean pressure, which in turn is higher than the right auricular mean pressure. The pulmonary capillary pressure may be elevated above the colloid osmotic pressure of the plasma during left ventricular failure, thus affording a mechanical explanation for pulmonary œdema. Mitral stenosis is indicated in the p.c.v. tracing by an abnormally high auricular pressure wave; mitral incompetence by an unusually early and high systolic pressure rise.

[Many tracings are reproduced relating the p.c.v. pressure waves with those in other parts of the heart and pulmonary circulation, and with phonocardiographic records. This article should be studied in the original by those interested.] *Martin Hynes*

The Effect of Body Position and Reference Level on the Determination of Venous and Right Auricular Pressure. J. O. DAVIES and N. W. SHOCK. *Amer. J. med. Sci.*, **218**, 281–290, 1949. 3 figs., 12 refs.

Ten subjects were studied; of these 3 were in congestive heart failure and one had a superior caval obstruction.

Technique is given in detail. [Those interested should consult the original paper.] There was no statistical difference between the recordings on the different instruments, nor did the venous pressure in the supine position differ significantly before and after tilting to the 45-degree position. Antecubital venous pressure increased with the assumption of a 45-degree sitting posture. The increase was from approximately 110 to 150 mm. saline. These results differ from those of Burch and his associates, who reported no such change. *A. T. Macqueen*

Comparison of the Constant and Instantaneous Injection Techniques for Determining Cardiac Output. W. T. RASHKIND and J. H. MORTON. *Amer. J. Physiol.*, **159**, 389–393, 1949. 3 figs., 5 refs.

Mechanism of the Auricular Arrhythmias. M. PRINZMETAL, E. CORDAY, I. C. BRILL, A. L. SELLERS, R. W. OBLATH, W. A. FLIEG, and H. E. KRUGER. *Circulation*, **1**, 241–245, 1950. 3 refs.

The mechanisms of the four auricular arrhythmias, namely, premature beats, paroxysmal tachycardia, auricular flutter, and auricular fibrillation, induced by the aconitine and post-electrical-stimulation methods, have been investigated in more than 200 dogs by means of high-speed cinematography, cathode-ray oscillography, and multiple-channel electrocardiography. Films of the auricles of the intact, exposed heart were taken in colour at speeds of 2000 frames a second, the motion of the auricles being slowed 250 times when the films were subsequently projected at 8 frames a second; auricular events that had originally occupied one second thus took 4 minutes to view on the screen. A magnifying lens was used which enlarged the image of the auricle 100 or more times on projection, and by means of such pictures the auricular contraction wave could be seen for the first time.

Auricular fibrillation was seen to be characterized by two phenomena: (1) Minute, irregular contractions which were continuously present. These were termed " M " contractions and involved an area of auricular wall approximately 0·03 to 3 mm. in diameter. (2) Large, rhythmic, wave-like contractions, termed " L " contractions, which swept across the auricle at a rate of 400 to 600 a minute, showing no sign of pursuing a circus path. In the ordinary electrocardiogram the familiar " f " waves probably represent the L contractions, and the M contractions are probably responsible for the tiny, rough irregularities on the " f " waves. It was concluded " that in both man and animals auricular fibrillation is a chaotic heterorhythmic disturbance," and that Lewis's hypothesis that a circus movement is present which gives rise to daughter waves is erroneous. Similarly, no evidence of a circus movement was found in the film records of auricular flutter, the flutter waves being similar to the L waves of auricular fibrillation, but more regular and generally more vigorous, originating at the point of local application of aconitine and spreading thence in all directions at once. The appearances in paroxysmal auricular tachycardia were essentially similar to those in auricular flutter, the only differences being (1) that the auricular rate in paroxysmal tachycardia was slower than in flutter and, consequently, each auricular wave was followed by a ventricular response (that is to say, there was no auriculo-ventricular block); and (2) in paroxysmal tachycardia the propagation of the individual waves was faster than that of the flutter waves.

The present conception of the action of anti-arrhythmic drugs such as quinidine and digitalis, namely, that they act largely through their supposed effect on the gap between the head and tail of the circus movement, becomes untenable in the light of these observations.

S. Oram

Auricular Fibrillation in Normal Hearts. H. H. HANSON and D. I. RUTLEDGE. *New Engl. Med.*, **240**, 947–953, 1949. 27 refs.

Auricular fibrillation is not always associated with organic heart disease, as was recognized as far back as 1911 by Mackenzie. The authors have reviewed 30 such cases, in patients who have been followed-up for periods ranging from 2 to 46 years, out of a total of 651 cases of auricular fibrillation seen at the Lahey Clinic, Boston, over a period of 20 years. In some of them the condition was paroxysmal and attacks might cease after some time; in others the condition remained persistent. It is pointed out that when the patient dies of other disease the heart shows no anatomical or histological abnormality. In ætiology, reflex autonomic activity would appear to play some part. The duration and frequency of paroxysms was unpredictable. If the patients survive sufficiently long the incidence among them of coronary disease, hypertensive heart disease, or other conditions is comparable to that in those of the population with previously normal hearts. There seems to be no

tendency to thrombus formation or subsequent embolism; the latter manifestation probably results from circulatory failure with dilatation of the auricles rather than auricular fibrillation without failure. This form of auricular fibrillation is probably much commoner than is generally recognized owing to the absence of symptoms.

In treatment the important thing is to watch the ventricular rate. As long as this remains at a reasonable level there is no need for any drug therapy. Quinidine may restore normal rhythm but the authors consider that even the very small risk of toxic effects may make its use undesirable. One patient in this series had been taking a maintenance dose of quinidine for 16 years and attacks still occurred whenever the drug was discontinued. *J. McMichael*

Congenital Malformations of the Heart. HELEN B. TAUSSIG, New York: The Commonwealth Fund. London: Geoffrey Cumberlege. (Pp. 618; 46 plates; 177 figs. £2 15*s*.)

With advancing experience in the special techniques of cardiac catheterization, gas analyses and angiocardiography, attention may be focussed away from the clinical aspect of diagnosis in congenital heart disease. But let this not detract from Dr. Taussig's book with its clear presentation and argument, the value of which is, indeed, enhanced by emphasis on the problem from the clinical angle. Her diagnostic interpretation of the deformed heart is made from the study of its functional capacity and anatomy, basing this on clinical and radioscopic assessment alone. The book is illustrated by excellent diagrams and X-rays which greatly simplify the text of her argument. Since her results are mainly derived from personal experience and observation, she admits to an incomplete bibliography. This will doubtless be remedied in her next edition, already inevitable, to embrace the immense advances in knowledge which surgical treatment has latterly brought about, and to satisfy the demand from those many workers in this field. *J. L. Lovibond*

Diseases of the Heart. CHARLES K. FRIEDBERG, M.D., Associate Physican, Mount Sinai Hospital, New York; Lecturer in Medicine, Columbia University. Philadelphia and London: W. B. Saunders Company. Price 57*s*. 6*d*.

This is a most complete modern textbook. It covers the recent advances in cardiology and emphasises the physiological background to clinical disorders of the heart. Special discussion is devoted to cardiac output, blood volume, extracellular fluids, blood oxygen concentration, intracardiac pressures and blood flow, and to demonstrating the growing practical importance of these quantitive measurements and their application to every day diagnosis and treatment in clinical cardiology. Circulatory failure, surgical advances, angiocardiography, and the newer knowledge of electrocardiography and other graphic methods such as electrokymography, are given full recognition and are lucidly described. The work is remarkable for its completeness and is noteworthy for the rich lists of references which follow each chapter. The exhaustive scientific and clinical literature has been most thoroughly covered and the bibliography is quite up-to-date. The volume will, therefore, serve a large field of readers; both teachers and students alike will benefit from its pages. The style is clear and concise and its presentation assimilable. Perhaps the radiograms could be bettered in subsequent editions.

J. L. Lovibond

A Manual of Cardiology. THOMAS J. DRY, Second Edition. Published by W. B. Saunders Company, Philadelphia and London. Price 25*s*.

The deserving popularity of this small book by the Associate Professor of Medicine, Minnesota University, is the basis for a second edition within seven years of its first appearance. The author's commendable clarity of description and the general arrangement of his text make it much more than a synopsis of the subject. In a work of this sort, it is difficult not to be dogmatic, indeed, the authority of some of his descriptions adds to their value, such as that of the thyrotoxic patient "who looks stimulated;" some readers might disagree with his statement that "the more effort it takes to produce anginal pain the better is the prognosis, regardless of the pathologic condition causing such coronary insufficiency." The chapter on congenital heart disease is clear and helpful, but in "the diagnostic features of the more important congenital anomalies" it is surprising to find a whole page devoted to mitral atresia, a rare condition. The subject of electrocardiography in relation to clinical problems is both comprehensive and readable, though his explanation of the pattern common in obesity is not one that is generally accepted. Emphasis on the fundamental considerations of the normal heart, and evidence of the writer's sane yet critical clinical balance, are prominent throughout this up-to-date book. *J. L. Lovibond*

Modification of the Cardiac Output after Intravenous Injection of Hypertonic Glucose Solution. M. SEGERS and J. P. WALSH. *Amer. J. med. Sci.*, **217**, 494–497, May, 1949.

A single intravenous injection of 50 ml. of hypertonic glucose solution (50%) was found to produce an increase in the cardiac output to 10 to 30% above the control figure in normal persons and those with cardiac disease. In normal subjects this augmentation was temporary; in the patients it was much more prolonged. The determinations of cardiac output were made by the ballistocardiographic method of Starr (*J. clin. Invest.*, 1940, **29**, 437). *A. I. Suchett-Kaye*

On the Mechanism by Which Intravenous Injections of Hypertonic Glucose Solution Cause Increased Cardiac Output. J. P. WALSH. *Amer. J. med. Sci.*, **217**, 498–504, May, 1949.

The results of experiments on normal subjects and patients with heart disease seem to show that increased filling of the heart is not the operating factor in increasing the cardiac output, since, although there was a significant

rise in circulating blood volume after injection of hypertonic glucose, the rise in venous pressure was " almost neligible." In the absence of evidence that other factors are involved, the author assumes that glucose has a specific stimulating action on the myocardium, as the rise in blood glucose level after injection could be constantly correlated with the increase in cardiac output. It was also noted that glucose had a more lasting effect on diseased than healthy cardiac muscle.

A. I. Suchett-Kaye

Treatment of Heart and Kidney Disease and of Hypertensive and Arteriosclerotic Vascular Disease with the Rice Diet. W. KEMPNER. *Ann. intern. Med.*, **31**, 821–856, Nov., 1949.

Benign hypertensive vascular disease can be effectively treated by the rice diet even when critical complications are present, and malignant hypertension may be greatly improved. Of 777 cases thus treated over 70% were benefited as judged by one of the following effects: decrease in the sum of systolic and diastolic pressures by at least 40 mm. Hg; reduction in heart diameter of 18% or more; correction of T_1 inversion; disappearance of severe retinopathy. Most of these patients were seriously ill and had failed to respond to other forms of treatment. The average time for a marked fall in blood pressure on the diet was three to four months. Of 520 patients, 286 had normal T waves in lead I and 102 abnormal ones before and after treatment; in 122 cases an inverted T_1 wave became upright and in 10 an upright one showed inversion during treatment. Chest radiography in 286 cases showed that 15 had an increase in heart diameter of about 3%, 146 had a decrease of about 6%, 106 had a decrease of about 14%, and 19 had a diminution in transverse diameter averaging 24%. The improvement in cardiographic pattern, the decrease in heart size, and the disappearance of papillœdema, hæmorrhages, and exudates in the fundi seem to bear little relation to fall in blood pressure and may occur on the diet without such a decrease.

The rice diet contains per 2000 calories, less than 5 g. of fat and about 20 g. of protein derived from rice and fruit and less than 200 mg. of chloride and 150 mg. of sodium. There is a marked decrease in the intake of nitrogen, sodium, chloride, and sulphate. Whereas in starvation and in protein deficiency the body uses its own protein with an increase in the blood level of non-protein nitrogen and urea nitrogen, on the rice diet urea and non-protein nitrogen levels decrease. Although salt intake is limited and serum chloride level does fall, restriction of protein in the diet outweighs this effect and protects against azotæmia. A high cholesterol level in the blood, which is often believed to be an important factor in vascular disease, almost invariably falls (by an average of 74 mg. per 100 ml. in 363 patients) on the rice diet.

T. Semple

Penicillin and Caronamide in Resistant Subacute Bacterial Endocarditis. C. H. STUART-HARRIS, J. COLQUHOUN, and J. W. BROWN. *Lancet*, **1**, 99–101, Jan. 15, 1949.

The authors describe 3 cases of subacute bacterial endocarditis in which combined treatment with caronamide and penicillin was effective, after treatment with penicillin alone had been followed by relapse in 2 cases—in one on 3 occasions. In 2 of the cases it was shown that the level of penicillin in the serum was considerably increased by the administration of caronamide and this probably was the factor leading to success of combined therapy. No serious toxic effects were observed but all the patients complained of nausea, and fever and leucopenia were observed in one; there was some sacral œdema in another, suggesting a temporary toxic effect on the renal function, though this has generally not been found in normal subjects.

Maurice Campbell

On the Mode of Formation of Lambl's Excrescences and their Relation to Chronic Thickening of the Mitral Valve. F. R. MAGAREY. *J. Path. Bact.*, **61**, 203–208, April, 1949.

Two hundred and fifty mitral valves were examined at necropsy in a series of cases from which only those of active rheumatic or bacterial endocarditis were excluded.

Lambl's excrescences were not found in subjects less than one year old and became increasingly common with age, being present in every patient over 60. This is a point against their being of congenital origin. On the mitral valve they occur singly or in groups on the auricular surface, chiefly along the line of closure. Shape varies, but they are usually fusiform and 5 or 10 mm. in length. They occur on apparently healthy valves, but are more numerous on vascularized and thickened cusps. Some of the fibrin deposits had lifted at one end and were undergoing organization. This is probably the way in which Lambl's excrescences are formed. Possibly the progressive organization of fibrin in rheumatic carditis leads to formation of rheumatic nodules on the cusps and later to stenosis of the mitral valve.

Peter Harvey

The Role of Desiccated Thyroid and Potassium Iodide in the Cholesterol-induced Atherosclerosis of the Chicken. D. DAUBER, L. HORLICK, and L. N. KATZ. *Amer. Heart J.*, **38**, 25–33, July, 1949. 1 fig., 20 refs.

A high-cholesterol diet in chickens raised their blood cholesterol level. Administration of desiccated thyroid tissue prevented this increase, but potassium iodide augmented it. The same diet induced atherosclerosis, but, whereas administration of desiccated thyroid tissue protected the chickens against this change, the action of potassium iodide was variable.

R. T. Grant

Production of Arteriosclerosis in Dogs by Cholesterol and Thiouracil Feeding. A. STEINER, F. E. KENDALL, and M. BEVANS. *Amer. Heart J.*, **38**, 34–42, July, 1949. 6 figs., 12 refs.

The serum cholesterol content in 3 dogs given thiouracil (1 g. daily) for over a year was doubled but necropsy showed no atherosclerosis. In 2 dogs given cholesterol (10 g. daily) for 16 months, the serum cholesterol level was trebled, but only minimal atherosclerosis in one case was found post mortem. However, in 2 dogs given both cholesterol and thiouracil, the serum cholesterol content was increased sixfold and eightfold respectively. At

necropsy both showed severe lesions which were very similar in distribution and in histology to those found in human atherosclerosis. *A. S. Dixon*

Plasma and Blood Infusion Following Myocardial Infarction. J. J. SAMPSON and I. M. SINGER. *Amer. Heart J.*, **38**, 54–68, July, 1949.

Acute myocardial infarction may be followed by a shock-like state of pallor, sweating, and hypotension, the severity of which is not related to the degree of myocardial infarction, or of congestive failure. The hypotension itself may be harmful in that it reduces irrigation of the remaining coronary bed and may produce relative anoxia of the brain or kidneys. Eleven patients with repeated myocardial infarction were given small blood or plasma transfusions on one or more occasions. Out of a total of 23 infarctions, 18 were treated by transfusion; in 11 cases the patient responded, but in 7 the patient died. Three other patients died suddenly after recovery from "shock," leaving one survival in the series. Response to transfusion was judged by the effect on blood pressure. There was evidence in some cases that further transfusion might have averted death; on the other hand the death of one patient, who had a high venous pressure, was probably hastened. *A. S. Dixon*

A Techinque for Division and Suture of the Patent Ductus Arteriosus in the Older Age Group. N. E. FREEMAN, F. H. LEEDS, and R. E. GARDNER. *Surgery*, **26**, 103–108, July, 1949.

Serious hæmorrhage during the dissection of a patent ductus arteriosus may be due to friability of the ductus wall, which is made more vulnerable by the difficulty of dissecting behind the duct. The latter is liable to be more than usually friable in older people owing to endarteritic changes.

Crafoord controls both pulmonary and systemic blood flow by placing clamps across the aorta and on the side of the pulmonary artery. This has the disadvantage that the distal aortic flow is prevented for a considerable time. Crafoord, however, reports no ill effects upon renal or other functions.

The present authors have employed a method which gives adequate control of both circulations without complete aortic occlusion, a modified Potts-Smith clamp being used to occlude the aortic end of the ductus. The pulmonary flow is controlled by digital pressure on the left pulmonary artery inside the pericardium. The method has been used with success on four dogs and one human subject. *W. P. Cleland*

The Hemodynamic Effects of Sympathectomy in Essential Hypertension. R. W. WILKINS, J. W. CULBERTSON, and M. H. HALPERIN. *Ann. intern. Med.*, **30**, 291–306, Feb., 1949.

The vascular and metabolic effects of surgical splanchnicectomy for hypertension were investigated in a series of Smithwick's own patients by means of direct arterial-pressure estimations with a Hamilton manometer, cardiac output determinations in which the Fick principle was applied and a cardiac catheter used, and hepato-portal blood-flow studies by the bromsulphalein method. When conditions were stabilized after operation, sympathectomy had not altered either the cardiac output or the blood flow to any important organ, which suggested a widespread decrease in peripheral resistance as the effective cause of the fall in blood pressure. The maintained renal blood flow may prove to be an adaptive response to lowered arterial pressure, and this problem is being studied further.

The decrease in splanchnic vasoconstrictor response in the erect position and the reduction of vasopressor overshoots of arterial pressure after sympathectomy were independent of the post-operative level of blood pressure, but the early increase in and later moderation of hepato-portal blood flow produced by sympathectomy was apparently directly related to the blood-pressure level. It is suggested that indirect physical or chemical mechanisms resulting from the direct hæmodynamic effects of splanchnicectomy may, in certain patients, act by producing a subsequent widespread decrease in peripheral resistance, thus leading to the fall in arterial pressure.
J. L. Lovibond

Transient Ventricular Fibrillation. II. The Effects of Gradually Induced Oxygen Deficiency on Patients with Transient Ventricular Fibrillation and on Patients with Periodic Standstill of the Ventricle. S. P. SCHWARTZ, G. C. LEINER, and R. J. MICHTOM. *Amer. Heart J.*, **37**, 918–926, May, 1949.

The effect of induced oxygen deficiency was studied in 2 patients suffering from transient attacks of ventricular fibrillation and in 2 patients with periodic standstill of the ventricles. The subjects rebreathed from a closed circuit until either loss of mental attention and sustained voluntary control, intense cyanosis, or abnormal changes in the type of breathing occurred.

In one patient in whom transient ventricular fibrillation developed during atrioventricular dissociation, progressive oxygen want easily converted a normal sinus rhythm into one of atrioventricular dissociation. In 2 patients further rebreathing during established atrioventricular dissociation increased the auricular rate, and short runs of ventricular fibrillation developed. In 2 patients, known to have syncopal attacks due to periodic standstill of the ventricles, rebreathing accelerated auricular rate and premature ventricular beats appeared. The appearance of these arrhythmias was irregular. It is suggested that anoxæmia is one of the factors responsible for the development of transient seizures of ventricular fibrillation in patients who are subject to such seizures during atrioventricular dissociation. *H. E. Holling*

Catheterization of the Coronary Sinus in Man. J. W. CULBERTSON, M. H. HALPERIN, and R. W. WILKINS. *Amer. Heart J.*, **37**, 942–951, May, 1949.

Four cases are recorded in which, during right heart catheterization, a cardiac catheter was inadvertently pushed into the coronary sinus. The procedure was watched on the X-ray screen, pressure tracings were taken

with a Hamilton manometer, and blood samples were analysed for oxygen.

The appearance of the catheter on the X-ray screen as it passes into the sinus is characteristic. The catheter, instead of passing from the right atrium to the left and downwards through the tricuspid valve moves directly to the left and upward in an oblique direction along the coronary sulcus. It always comes to a full stop at the left border of the heart, and, so long as it is in the sinus, it always follows exactly the same course. The dark venous colour of the blood withdrawn from the sinus is characteristic. The average oxyhæmoglobin saturation of coronary-sinus blood in these 4 cases was found to be 30%, whereas that of mixed venous blood (50 subjects) was found to be 73%. Pressure tracings showed that the mean pressure in the sinus was from 0 to 15 mm. Hg; the tracings differed from those obtained in the atrium in that the curves ranged slightly higher and showed three waves of vibrations instead of the characteristic atrial pulses. *H. E. Holling*

Kymographic Demonstration of the Systolic Expansion of the Left Auricle in Mitral Disease. D. ROUTIER, R. HEIM DE BALSAC, and L. ALESSANDRIS. *Sem. Hôp. Paris*, **26**, 60–71, Jan. 6, 1950.

The authors describe the use of radioscopy and kymography in the demonstration of systolic expansion of the left auricle in 190 cases of mitral disease. They attribute the auricular expansion to regurgitation of blood during ventricular contraction.

The systolic auricular expansion was particularly marked in cases of arrhythmia and in the presence of ventricular enlargement, and was observed both in the presence of a systolic bruit and in cases of clinically pure mitral stenosis. On the other hand, a systolic bruit might be present without demonstrable auricular expansion, the absence of which does not, therefore, exclude mitral regurgitation, which may be insufficient to dilate the auricle, but nevertheless be sufficient to cause a vibration of the valves which manifests itself in the bruit. However, the extent of the auricular expansion when present provides a rough indication of the volume of blood regurgitated and may thus serve as a guide in the assessment of progress. *A. Orley*

Ætiology and Treatment of Auricular Flutter. D. H. MAKINSON and G. WADE. *Lancet*, **1**, 105–108, Jan. 21, 1950.

Over a 5-year period, 16 cases of auricular flutter were found in a total of 9458 patients subjected to electrocardiographic examination. Careful exclusion of cases of "impure flutter," in which the rhythm was not strictly regular may be responsible for the low incidence of this irregularity as compared with other published series. For purposes of analysis of aetiology, a number of additional cases from private practice were included, making a total of 36 cases, of which 14 were classified as rheumatic in origin, 13 as degenerative heart disease, and 9 as "miscellaneous" in origin including doubtful cases of rheumatism (2), cases of congenital heart disease (2), convalescence from severe illness (2), thyrotoxicosis (1), and rheumatoid arthritis (1), and one case in which no other evidence of disease was detected. The incidence was greatest in the fifth and sixth decades, and before this age rheumatism was the dominant causative factor. There were 29 males to 7 females, this sex distribution being ascribed to the special liability of the male to degenerative cardiovascular disease.

Digitalis was used alone in treatment in 14 cases, normal rhythm being induced in 8 (relapsing subsequently in 2), fibrillation supervening in 4, and flutter persisting in 2. In 9 cases quinidine was given after initial treatment with digitalis, but restored normal rhythm in only 1 of the 4 cases in which fibrillation had been induced and in none of those in which flutter persisted. Quinidine alone restored normal rhythm in 1 of 3 cases thus treated. In those cases which reverted to fibrillation on digitalis therapy there was a close correlation between the time required to induce fibrillation and the dosage of digitalis: 3 grains (0·2 g.) of the powdered leaf thrice a day induced a change of rhythm within 4 days, but if only 2 or 3 grains was given daily periods of treatment of up to 3 months were required. The waves of auricular activity in flutter are best demonstrated electrocardiographically in lead II or lead III, but sometimes better records are obtained with leads from the right præcordium. *J. McMichael*

Acute Serofibrinous Pericarditis of Undetermined Cause. A Study of Twenty-seven Cases. R. L. LEVY and M. C. PATTERSON. *Amer. J. Med.*, **8**, 34–45, Jan., 1950.

The authors describe 27 cases of acute pericarditis of obscure ætiology encountered during a period of 15 years. In 23 there was a history of preceding respiratory tract infection, and it is suggested that these conditions may have been due to virus infection. In other cases the pericarditis was thought to be an allergic reaction to bacterial infection of the throat or nasal sinuses.

The course of the attack varied but all patients recovered completely without sequelæ. Follow-up examinations 6 months to 15 years later revealed no evidence of heart disease in any case. The differential diagnosis from tuberculous and rheumatic pericarditis, and from pericarditis accompanying myocardial infarction, is discussed. Electrocardiography is of assistance in distinguishing the last condition. It is noteworthy that the prolongation of the P–R interval so often seen in acute rheumatism was never observed.

Treatment with sulphonamides, penicillin, and streptomycin seemed to exert no appreciable effect on the course of the disease. The authors suggest that aureomycin treatment would be worth trying. *John Forbes*